Causes of Mortality in Sea Ducks (Mergini) Necropsied at the USGS-National Wildlife Health Center

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Abstract.—A number of factors were identified as causes of mortality in 254 (59%) of 431 sea ducks submitted for necropsy at the USGS-National Wildlife Health Center, Madison, Wisconsin from 1975 until 2003. Bacteria causing large outbreaks of mortality were *Pasteurella multocida* and *Clostridium botulinum* Type E. Starvation was responsible for large mortality events as well as sporadic deaths of individuals. Lead toxicity, gunshot and exposure to petroleum were important anthropogenic factors. Other factors that caused mortality were avian pox virus, bacteria (*Clostridium botulinum* Type C, *Riemerella anatipestifer* and *Clostridium perfringens*), fungi (*Aspergillus fumigatus* and an unidentified fungus), protozoans (unidentified coccidia), nematodes (*Eustrongylides* spp.), trematodes (*Sphaeridiotrema globulus* and *Schistosoma* spp.), acanthocephalans (*Polymorphus* spp.), predation, cyanide and trauma (probably due to collisions). There were also a number of novel infectious organisms in free-living sea ducks in North America, which were incidental to the death, including avipoxvirus and reovirus, bacteria *Mycobacterium avium*, protozoans *Sarcocystis* sp. and nematodes *Streptocara* sp. Apart from anthropogenic factors, the other important mortality factors listed here have not been studied as possible causes for the decline of sea ducks in North America. *Received 24 April 2004, accepted 22 January 2005.*

Key words.—Sea ducks, *Mergini*, mortality, disease, *Pasteurella multocida*, *Clostridium botulinum*, starvation, lead toxicity, firearm, petroleum.

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Sea ducks (Mergini) have declined generally over the past 20 years in North America with some, such as the eastern population of Harlequin Duck (*Histrionicus histrionicus*), the Spectacled Eider (*Somateria fischeri*) and the Steller's Eider (*Polysticta stelleri*), currently being listed as endangered or threatened (Sea Duck Joint Venture Management Board 2001). Despite the decline of ten of the 15 species of sea ducks in North America, very few causes have been identified (Sea Duck Joint Venture Management Board 2001). Outside of North America, Common Eider (*Somateria mollissima*) numbers in the Baltic Sea have declined by 50% due to increased juvenile mortality, possibly due to a reovirus (Hario 1998; Hollmén *et al.* 2002). One ready method of determining likely causes of mortality is necropsy of sick and dead animals. This is standard practice for domestic animals worldwide and has been more recently used as a standard procedure for wild animals in the United States of America (USA) (Davidson and Nettles 1997; Friend and Franson 1999). The value of necropsy in diagnosing factors causing mortality in declining free-living animals is perhaps best illustrated by the discovery of a novel pathogen, *Batrachochytrium dendrobatidis*, causing the decline and sometimes extinction of wild amphibian species in Australia, Latin America, North America, and Spain (Berger *et al.* 1998; Bosch *et al.* 2000; Bradley *et al.* 2002; Green *et al.* 2002). Here, we report causes of mortality in sea ducks derived from necropsy findings from 1975 through 2003 at the USGS-National Wildlife Health Center (NWHC) of the USA and identify factors that may contribute to population declines.

METHODS

We reviewed the NWHC database from 1975-2003 for causes of death and novel information pertaining to sea ducks. The database summarizes necropsy records

for carcasses submitted to NWHC for diagnostic evaluation and epizootic information for mortality events in wildlife in the USA, respectively (see NWHC 2004a, 2004b for metadata). Guidelines for specimen collection, preservation and shipment were provided to submitters so that post mortem change was minimized (Friend and Franson 1999). These observations were made opportunistically rather than systematically, and therefore may not be representative of the important causes of mortality within sea ducks over time. Complete hard copy files were checked for accuracy of the electronic data and to gather additional information on a subset of records for which there was an etiological diagnosis or a novel finding. Only wild birds for which an attempt was made to diagnose the cause of mortality (N = 431) are included here. In addition to NWHC diagnostic cases, we reviewed necropsy and laboratory findings from our research studies on sea ducks and included two novel incidental findings. Estimates of the total mortality in the population are also provided when available.

Necropsy procedures at the NWHC included an external and internal gross examination to evaluate organ systems and identify abnormalities. Pathologists used history and gross observations to determine the need for additional laboratory testing of tissues and organs including histopathology, culture for bacteria, viruses and fungi, examination for parasites and chemical analyses for toxins. For 90% of birds, gross necropsy included one or more additional diagnostic tests. The selective use of diagnostic tests based on a priori information is a widely used technique in human and veterinary medicine in order to make investigations of disease cost effective. However, this technique is more likely to lead to failure of detection of diseases compared with systematic testing. Therefore, negative diagnostic findings should be interpreted with caution and are not reported in detail here. Histopathology was the most common test used to supplement gross observations seen at necropsy and was usually routinely performed on the major tissues including brain, heart, lung, liver, kidney, spleen, bursa when present, intestine and pancreas. In general, when changes in the size, texture, or color pattern were observed in organs, they were cultured for fungi, bacteria and viruses. Polymerase chain reaction tests that look for genetic fragments of *Chlamydiophila psitacci* and viruses (e.g., West Nile virus, avian influenza and duck plague virus) in tissues have been utilized in recent years in addition to culturing. Toxins such as heavy metals, pesticides and avian botulinum toxin types C and E were also considered when birds were in good body condition with no obvious cause of death or had lesions suggestive of toxicity such as streaks of pallor in the heart or liver fibrosis. Tissues for histopathology were fixed in 10% buffered formalin, processed for light microscopy, and stained with haematoxylin and eosin for routine examination and special stains were used as appropriate. For bacterial isolation, tissues were inoculated onto 5% sheep red blood cell agar and eosin-methylene blue plates (DIFCO®, Becton Dickinson, Sparks, MD), incubated at 37°C for 48h, and characterized by one or more of the following systems: Crystal® (Becton Dickinson, Sparks, MD); MicroPlate® (Biolog, Hayward, CA); API®, VITEK® (bioMéreux, Hazelwood, MO). Blood collected from the hearts of sea ducks was used to test for toxins of *Clostridium botulinum* types C and E with the mouse neutralization test (Quortrup and Sudheimer 1943). Blood was centrifuged and the serum was harvested and inoculated into mice receiving antitoxin specific for botulinum types C and E, and mice receiving no antitoxin. The mice were observed for 5 to 7 days for clinical signs of botulism. Botulism type C or E was diagnosed when toxin was demonstrated in heart blood serum (unprotected mice developed clinical signs or died and the mice receiving the corresponding antitoxin survived), carcasses were in good to fair post mortem condition and cause of death could be attributed to no other finding. A diagnosis of avian botulism was also frequently corroborated by the finding of a number of other dead waterfowl in the same vicinity. A diagnosis of suspect botulism was assigned when specimens were in poor post mortem condition or when the post mortem condition was not recorded. This suspect classification was also applied to other bacterial pathogens, which may invade and/or multiply in a carcass post mortem such as *Clostridium perfringens*. Tissues for virus isolation were processed for inoculation into Muscovy Duck (*Carina moschata*) fibroblasts (MSDEF) (Docherty and Slota 1988) and embryonating chicken eggs (Senne 1998) as described by Hollmén *et al.* (2002). These methods are effective in isolating known important viral pathogens of birds. Briefly, tissues (approximately 1 g) were homogenized in viral transport medium, homogenates were centrifuged, and supernatants were inoculated into MSDEF monolayers and into the chorioallantoic chamber of embryonating chicken eggs. These eggs were monitored for mortality and MSDEF cell cultures were examined microscopically for viral cytopathic effects. Identification of avian pox was based on the presence of inclusion bodies in the chorioallantoic membrane from inoculated chicken eggs.

Lead concentrations in samples of liver were determined at the Wisconsin Department of Agriculture, Central Animal Health Laboratory, Madison, Wisconsin from 1980 through 1984 according to Boyer (1984), and at NWHC according to Locke *et al.* (1991) from 1985-1991 and Franson and Smith (1999) from 1992 onwards. Lead poisoning was diagnosed when one or more characteristic lesions of lead poisoning (Beyer *et al.* 1998) were present, combined with a liver lead concentration of >6 ppm, wet weight (Pain 1996). When pesticide poisoning was suspected based on a history of possible exposure, brain acetylcholinesterase activity was measured colorimetrically at NWHC and interpreted according to Smith *et al.* (1995), and gastrointestinal contents were analyzed for organophosphorus and carbamate compounds at the Patuxent Analytical Control Facility, Laurel, Maryland (Smith *et al.* 1995). When field history suggested cyanide poisoning (carcasses found on or near cyanide-treated mine tailings ponds), brain and/or blood was analyzed by microdiffusion analysis (Feldstein and Klendshoj 1957). The lower limit of detection was 0.03 ppm cyanide on a wet weight basis. Cyanide poisoning was diagnosed when cyanide concentrations were >0.05 ppm in brain tissue or >1 ppm in blood, wet weight (Ballantyne and Marrs 1987). Because testing for marine biotoxins has only recently become available and interpretation of their presence in tissues of birds is still often unclear, this was not one of the diagnostic tests applied to the birds in this study.

Starvation was diagnosed in emaciated carcasses when no injuries, lesions or other factors, which may have prevented the bird from obtaining adequate food, were identified. It is possible that some factors such as viruses, which may have predisposed birds to starvation, were occasionally overlooked given that viral culture was not carried out systematically. Predation was diag-

nosed when there was evidence of antemortem blood loss and infusion of blood into tissues associated with findings such as presumptive bite marks, partial consumption of the carcass, and a history indicating no other probable source of traumatic death. A few birds had more than one disease contributing significantly to death. For example, starvation was seen with diseases such as lead poisoning or peritonitis due to acanthocephalans. When more than one etiology occurred, they were recorded separately. There were a number of necropsies where a disease, which caused the death of the animal, was identified, but the etiology could not be confirmed or determined. Autolysis prevented determination of an etiology for mortality for some birds.

RESULTS

Of the 431 sea ducks that were necropsied, the etiology for cause of death was determined for 254 birds (59%) (Table 1). The most common species for which an etiology was identified were Common Eider, White-winged Scoter *(Melanitta fusca*), Surf Scoter (*M. perspicillata*), Long-tailed Duck *(Clangula hyemalis*), Common Goldeneye *(Bucephala clangula*), Bufflehead *(B. albeola*), Red-breasted Merganser (*Mergus serrator*), and Common Merganser *(M. merganser*). Species for which fewer than ten individuals were examined were King Eider (*Somateria spectabilis*), Steller's Eider, Black Scoter (*Melanitta nigra*), Harlequin Duck, Barrow's Goldeneye (*Bucephala islandica*) and Hooded Merganser (*Lophodytes cucullatus*). Sick and dead sea ducks were submitted from 35 states. Half of the sea ducks came from states on the west coast with the largest number being submitted from Alaska. Table 2 lists diseases and parasites that resulted in death and Table 3 lists anthropogenic factors such as lead poisoning and gunshot. Starvation, trauma and predation are listed as other factors in Table 3. In addition, twelve birds that collided with man-made structures were classified as dying from trauma and listed with other birds that died from trauma under other factors in Table 3 and not under anthropogenic factors.

Biotic Factors Causing Mortality

Large-scale mortality was caused by the bacterium *Pasteurella multocida*, which causes avian cholera (Table 2). Large-scale mortality due to avian cholera occurred primarily in Long-tailed Ducks in Chesapeake Bay in the winters of 1978 and 1994. Small numbers of White-winged Scoter were also affected in 1978 as well as other species of sea ducks (reported in Montgomery *et al.* 1979). More frequent moderate-scale mortality due to avian cholera occurred in the southern race of the

Table 1. Numbers of sea ducks submitted to the USGS-National Wildlife Health Center, Madison, Wisconsin, USA from 1975-2003 and etiologies for disease leading to death.

Species	Total of etiologies	No etiology	Total
Common Eider	46	6	52
Spectacled Eider	7	13	20
King Eider	2	1	3
Steller's Eider	1	0	1
White-winged Scoter	33	13	46
Surf Scoter	22	17	39
Black Scoter	5	1	6
Unidentified scoter	0	1	1
Long-tailed Duck	24	28	52
Harlequin Duck	1	4	5
Common Goldeneye	37	29	66
Barrow's Goldeneye	1	1	2
Unidentified goldeneye	2	11	13
Bufflehead	25	24	49
Red-breasted Merganser	13	16	29
Common Merganser	22	5	27
Hooded Merganser	8	0	8
Unidentified merganser	5	7	12
Total	254	177	431

Table 2. Biotic factors causing mortality in sea ducks submitted to the USGS-National Wildlife Health Center, Madison, Wisconsin, USA from 1975-2003.

	Species										
Etiology	Common Eider	White winged Scoter	Surf Scoter	Long-tailed Duck	Common Goldeneye	Buffle-head	Red-breasted Merganser	Common Merganser	Hooded Merganser	Unidenti-fied Merganser	Total
Virus avian pox (tracheitis)					1[a]						1
Bacterium *Pasteurella multocida* (avian cholera)	12	2		12[b]	5	4		1		1	37
Bacterium *Clostridium botulinum* Type E (botulism)		1		1	3		4				9
Bacterium *Clostridium botulinum* Type C (botulism)					8[c]	2			1		11
Bacterium *Riemerella anatipestifer* (septicaemia)		1									1
Bacterium *Clostridium perfringens* (necrotic enteritis)		1									1
Fungus *Aspergillus fumigatus* (disseminated mycosis)	1					1		2			4
Fungus (encephalitis)							1				1
Coccidium (enteritis, typhlitis)	1	1									2
Nematode *Eustrongylides* spp. (peritonitis)		1						1	1		3
Trematode *Schistosoma* sp. (enteritis)		2									2
Trematode *Schistosoma* sp. (encephalitis)					1						1
Trematode *Sphaeridiotrema globulus* (haemorrhagic enteritis)						2					2
Acanthocephalan *Polymorphus* spp. (peritonitis)	2		7								9

[a]Recorded in Hansen (1999).
[b]Some of these birds were reported in Montgomery (1979).
[c]The post mortem condition of these animals was not recorded and therefore the diagnosis is suspect.

Table 3. Anthropogenic and other factors causing mortality in sea ducks submitted to the USGS-National Wildlife Health Center, Madison, Wisconsin, USA from 1975-2003.

	Species[a]																	
Etiology	COEI	SPEI	KIEI	STEI	WWSC	SUSC	BLSC	LTDU	HARD	COGO	BAGO	UGO	BUFF	RBME	COME	HOME	UME	Total
Anthropogenic factors																		
Lead (toxicity)	1[b]	5[b]								11[c]					2		1	20
Petroleum (toxicity)	1					3	1	1	1						3	3	1	14
Cyanide (toxicity)													15					15
Firearm (trauma)	4	2		2						1		2	7		10	3		31
Fishing net (drowning)	3																	3
Other factors																		
Starvation (emaciation)	15		1		23	17	3	1		4	1		2	6		1	2	76
Trauma	11		1	1	1	2		7		3			3	2	3			34
Predation							1	2										3

[a]COEI Common Eider, SPEI Spectacled Eider, KIEI King Eider, STEI Steller's Eider, WWSC White-winged Scoter, SUSC Surf Scoter, BLSC Black Scoter, LTDU Long-tailed Duck, HARD Harlequin Duck, COGO Common Goldeneye, BAGO Barrow's Goldeneye, UGO unidentified goldeneye, BUFF Bufflehead, RBME Red-breasted Merganser, COME Common Merganser, HOME Hooded Merganser, UME unidentified merganser.

[b]Five of these birds were reported in Franson *et al.* (1995).

[c]Three of these birds were reported in Beyer *et al.* (1998).

Common Eider (subspecies *dresseri*), predominantly females, on breeding grounds off the Maine coast. *Pasteurella multocida* serotype 3 × 4 was most commonly isolated from cases of avian cholera in the Atlantic flyway (Table 4). In contrast, serotype 1 caused mortality in the Mississippi, Central and Pacific flyways. Mortality in these flyways occurred predominantly in waterfowl other than sea ducks. If sea ducks were affected in the Mississippi, Central and Pacific flyways, they comprised <5% of the birds affected and were often not submitted to the NWHC.

Mortality due to avian botulism produced by *Clostridium botulinum* type E was diagnosed in birds from the Great Lakes and Alaska from 1999 to 2003 (Table 2). Avian botulism due to *C. botulinum* type C occurred in three different species of sea ducks with few individuals submitted to the NWHC (Table 5). Mortality due to *C. botulinum* type C occurred predominantly in waterfowl other than sea ducks with <5% of the birds affected being sea ducks. For this reason, sea ducks were often not submitted to the NWHC during avian botulism Type C epizootics.

The next most frequent infectious pathogens were the acanthocephalan parasites *Polymorphus* spp. causing peritonitis by burrowing through the intestinal wall, and the fungus *Aspergillus fumigatus* causing disseminated granulomata. Heavy burdens of *Polymorphus altmani* (719-2,224 adult worms per bird) causing peritonitis and possibly emaci-

Table 4. Estimated sea duck mortalities due to avian cholera and number of other avian species killed in outbreak. Estimates derived from collection and necropsy of a sample of birds by the USGS-National Wildlife Health Center, Madison, Wisconsin, USA from 1975-2003.

Species	Mortality	Location	States	Date	Period	Serotype	No. of other species killed
Long-tailed Duck	>25,000[a]	Chesapeake Bay[b]	MD, VA	Mar. 1978	Wintering	3, 3 × 4	18
	>31,500[c]	Chesapeake Bay	MD, VA	Feb. 1994	Wintering	3 × 4	57
Common Eider	2,500	14 islands, Blue Hill Bay	ME	June 1980	Breeding	3, 3 × 4	3
(subspecies *dresseri*)	Hundreds	12 islands, Muscongus Bay	ME	June 1984	Breeding	1	3
	20	Petit Manan & Green Islands	ME	June 1985	Breeding	3	0
	200	Little Birch & Stockman Islands	ME	May 1987	Breeding	3 × 4	2
	46	Popham	ME	June 1989	Breeding	4	0
White-winged Scoter	>344	Chesapeake Bay	MD, VA	Mar. 1978	Wintering	3 × 4	18
	1	Cook Inlet	AK	Nov. 1988	Migrating	NA[d]	0
Common Goldeneye	>2	Freezeout Game Mgmt. Area	MT	Sept. 1977	Migrating	NA	2
	1	Cottonwood Lake	SD	Mar. 1980	Migrating	NA	1
	1	Tule Lake NWR[e]	CA	Dec. 1985	Wintering	1	11
	1	Lac Qui Parle WMA[f]	MN	Nov. 1991	Migrating	1	5
Bufflehead	2	Tule Lake NWR	CA	April 1976	Migrating	1	3
	1	Tule Lake NWR	CA	Oct. 1977	Migrating	1	4
	1	Lake Ewauna, Veterans Park	OR	Jan. 1998	Wintering	NA	2
Common Merganser	1	Harvard WPA[g]	NE	Feb. 1991	Wintering	1	5
Mergus sp.	1	Tule Lake NWR	CA	Dec. 1977	Wintering	1	5

[a]Reported in Montgomery (1979).
[b]Mortality occurred on Chesapeake Bay from Baltimore county, Maryland south to Norfolk, Virginia and on the Atlantic Ocean from the entrance to Chesapeake Bay to the North Carolina line.
[c]Referred to in Hindman 1997.
[d]NA Not Available.
[e]NWR National Wildlife Refuge.
[f]WMA Wildlife Management Area.
[g]WPA Waterfowl Production Area.

Table 5. Estimated sea duck mortalities due to avian botulism and number of other avian species killed as determined by the USGS-National Wildlife Health Center, Madison, Wisconsin, USA from 1975-2003.

Species	Mortality	Location	State	Date	Period	Type of botulism	No. other avian species killed
Common Goldeneye	12	Upper Mississippi NWR	WI	April 1977	Migrating	C	1
Hooded Merganser	1	McCarthy Park, Milwaukee	WI	Aug. 1984	Breeding	C	2
Bufflehead	1	Farwell	TX	Nov. 1980	Migrating	C	1
	1	City Park, Denver	CO	Aug. 1986	Breeding	C	>9
Common Goldeneye	1	Haines	AK	Sept. 1999	Migrating	E	1
Red-breasted Merganser	45	Presque Isle State Park, Lake Erie	PA	Dec. 2000	Wintering	E	6
Long-tailed Duck	10	Presque Isle State Park, Lake Erie	PA	Nov. 2001	Migrating	E	11
Common Goldeneye[a]	3	Lake Ontario	NY	Nov. 2003	Migrating	E	2
White-winged Scoter[a]	1	Lake Ontario	NY	Nov. 2003	Migrating	E	2

[a]Same mortality event.

ation in seven Surf Scoter were associated with an estimated mortality of 100 Surf Scoter along the coast of California around San Francisco in the spring of 1995. *Polymorphus* sp. causing peritonitis was diagnosed in two birds and emaciation in six others that were submitted from an estimated mortality of 1,000 and at least 30 Common Eider at Cape Cod, Massachusetts in the springs of 1998 and 2000, respectively.

The poxvirus and the remaining bacteria, fungi and parasites listed in Table 2 were diagnosed as causes of mortality in one or two individuals. Two White-winged Scoter that were killed by a schistosome were from a large mortality event that killed at least 142 scoters at Cape Suckling, Alaska in August 1992. Two other of the four birds submitted from this mortality event died from emaciation rather than schistosomiasis.

Anthropogenic and Other Factors Causing Mortality

Emaciation was the most frequent diagnosis, and was assumed to be the result of starvation when no other causes were identified (Table 3). In some cases the mortality event due to starvation was large. For example, 5,000 White-winged Scoter and Surf Scoter and 1,000 of all three species of scoter were estimated to have died from emaciation at Cape Yakataga, Alaska in August 1990 and 1991, respectively, although only 28 in total were submitted for necropsy. In November 1984, 150 White-winged Scoter and Surf Scoter died at Bodega Bay, California. Fifty-two Red-breasted Merganser died from emaciation at Nantucket Island, Massachusetts in March 1986 and at least 76 Common Eider died from emaciation at Cape Cod Bay, Massachusetts in June 1991. Firearm trauma and non-specific trauma, probably the result of collisions with various types of structures during periods of poor visibility, were diagnosed in 31 and 34 sea ducks, respectively. Drowning from entanglement in fishing nets was diagnosed in three Common Eiders. Lead poisoning was the most frequently identified toxicosis, occurring in 20 individuals of four species. Mean ± SD (min-max) liver lead concentrations were 31.8 ± 14.3 ppm wet weight (12.0-65.9). An ingested lead sinker (4 × 11 mm) was recovered from the stomach of a Common Merganser found dead in Idaho in 1997. Ingested lead shot were also found in a Spectacled Eider (*Somateria fischeri*) from Alaska in 2000. Previously published reports of lead poisoning in sea ducks examined at NWHC include three Common Goldeneye (one with ingested lead shot) from Montana in 1984 (Beyer *et al.* 1998) and one Common Eider and four Spectacled Eider (the Common Eider and one of the Spectacled Eiders had ingested lead shot) found in Alaska in 1992 and 1993, respectively (Franson *et al.*

1995). One Common Goldeneye was probably poisoned by ingesting sediment from the Coeur d'Alene River Basin, Idaho, which is contaminated with lead from mine tailings (reported in Sileo *et al.* 2001). Four Bufflehead that died of cyanide poisoning were found in association with four gold-mine tailings ponds that contained cyanide in California, Idaho, Nevada and Washington. Oiling from petroleum spills was diagnosed in three Common Merganser from Wyoming in 1980, three Surf Scoter from California in 1990 (although at least 120 died from the spill), a Common Eider from Rhode Island in 1996 (although several more individuals and other species of sea ducks were affected in the spill) and one Hooded Merganser from Oregon in 2001. In addition, one Black Scoter, one Harlequin Duck, and one Long-tailed Duck were submitted from Prince William Sound, Alaska, after the Exxon Valdez oil spill in 1989, which has been estimated to have killed between 100,000 and 690,000 sea birds (Piatt and Ford 1996). Oiling was also diagnosed in two Hooded Merganser found near an oil sludge pit in Pennsylvania in 1981, and in one unidentified merganser found near an oil pit in Oklahoma in 1990. Sea ducks and other species were killed by an oil spill in New Jersey in 1990, but no carcasses of sea ducks were submitted for necropsy.

There were a number of novel incidental findings including avian pox virus that caused bumblefoot type lesions in a Harlequin Duck (also reported in Hansen 1999), a reovirus in a Long-tailed Duck and disseminated *Mycobacterium avium* abscesses in a Long-tailed Duck that died from colliding with an oil facility structure in the Beaufort Sea near Prudhoe Bay, Alaska. There were also *Sarcocystis* sp. in the pectoral muscles of a Black Scoter and two Steller's Eider, an unidentified protozoan that caused cholecystitis in a Spectacled Eider and the nematode parasite *Streptocara* sp. causing ulcerative proventriculitis in a Common Goldeneye. These organisms did not cause significant morbidity apart from *M. avium* which probably caused the severe emaciation seen in the Long-Tailed Duck and would have eventually killed the bird, and the *Streptocara* sp. which probably caused the poor body condition of the Common Goldeneye. A newly recognized disease of unknown etiology, avian vacuolar myelinopathy, was diagnosed in two Buffleheads in central North Carolina (see Augspurger *et al.* 2003). An incidental finding of an intramuscular lipoma was seen in a Long-tailed Duck.

DISCUSSION

Avian cholera is an important disease of sea ducks along the Atlantic flyway that has affected mainly wintering Long-tailed Duck and breeding Common Eider. In addition to the mortality reported here, there was large mortality of wintering Long-tailed Duck (5,441 carcasses collected) in Chesapeake Bay in 1970 due to avian cholera (Locke *et al.* 1970). During these large mortality events in Long-tailed Ducks, other sea ducks including all three species of scoter, Common Goldeneye, Bufflehead and Red-breasted Merganser, also died of avian cholera (Locke *et al.* 1970; Montgomery *et al.* 1979, this study). For example, 1,580 White-winged Scoter carcasses were collected during the mortality event in 1970 (Locke *et al.* 1970). Mortality of the southern race Common Eider from avian cholera has been observed intermittently on nesting grounds off the Maine coast and in the St. Lawrence estuary (Gershman *et al.* 1964; Reed and Cousineau 1967; Reed 1975; Korschgen *et al.* 1978; Wobeser 1994; Giroux *et al.* 2002). Avian cholera is also an important disease of the Common Eider in Scandinavia (Swennen and Smit 1991; Christensen *et al.* 1997; Pedersen *et al.* 2003). *Pasteurella multocida* serotypes 3, 3 × 4 or 4 were most commonly isolated, followed by serotypes 4 × 12, 3 × 12 and 1 when sea ducks are the primary species affected along the Atlantic flyway and in Europe. Serotype 1 predominated in other waterfowl in the Mississippi, Central and Pacific flyways (Locke *et al.* 1970; Heddleston *et al.* 1972; Korschgen *et al.* 1978; Montgomery *et al.* 1979; Brogden and Rhoades 1983; Hindman *et al.* 1997; Christensen *et al.* 1998; Friend 1999a; this study). Avian cholera occurred in few sea ducks in the Mississippi, Central and

Pacific flyways of North America (Zinkl *et al.* 1977; this study). An outbreak of avian cholera was recently diagnosed in the Common Eider in Denmark (Christensen *et al.* 1997). This disease has the potential to continue to emerge in sea ducks. Despite the importance of avian cholera in sea ducks, there has been no broad systematic study of this disease in this group of waterfowl.

The occurrence of avian botulism due to *C. botulinum* type E in sea ducks in the Great Lakes is of concern. Previously, this disease has been reported primarily in loons and gulls in that area (Brand *et al.* 1983, 1988), In addition to the mortality reported here, large scale botulism type E mortality in the Common Merganser and Red-breasted Merganser occurred in autumn 1999 and 2001 on Lakes Huron and Erie and in the Long-tailed Duck in autumn 2002 on Lakes Erie and Ontario (Canadian Cooperative Wildlife Health Centre unpublished observations; New York Department of Environmental Conservation unpublished observations). The reasons for the emergence of this disease in the Great Lakes may be difficult to determine, given the variety and complex interactive nature of environmental conditions that affect type C avian botulism outbreaks (Rocke and Samuel 1999). The occurrence of type E avian botulism in an unidentified goldeneye in Alaska is also a warning of the potential expansion or presence of this disease in other areas. Avian botulism due to *C. botulinum* type C is one of the most important diseases of waterfowl but, because it occurs predominately in filter feeders and dabblers, sea ducks are not commonly affected (Rocke and Friend 1999). However, recent large-scale mortality of the White Pelican (*Pelicanus erythrorhynchos*) and Brown Pelican (*P. occidentalis*) due to this disease suggests that avian botulism type C could have a greater affect on fish eating birds than previously thought (Friend *et al.* 2001; Friend 2002).

Of the other pathogens causing mortality, *Aspergillus fumigatus*, *Riemerella anatipestifer*, *Sphaeridiotrema globulus*, *Polymorphus* spp., *Eustrongylides* spp. and schistosomes have been reported in waterfowl (Wobeser 1997). In sea ducks, *Eustrongylides tubifex* has caused significant mortality in the Red-breasted Merganser in December in Virginia (Cole 1999) and *Polymorphus* spp. (syn. *Profilicollis*) have killed Common Eiders in Europe (Clark *et al.* 1958; Swennen and Van den Broek 1960; Rayski and Garden 1961; Grenquist 1970; Persson 1974; Mörner 1978; Itämies *et al.* 1980). Attributing mortality to *Polymorphus* spp. should be based on the associated pathology rather than the number of worms, since sea ducks may harbor in excess of 1,000 with no obvious clinical ill effect (Wobeser 1997; Camphuysen *et al.* 2002). No systematic study has assessed whether high intensities of infection and the pathology induced by the acanthocephalans are a primary cause of starvation or secondary to reduced food availability. Birds may switch to heavily parasitized, non-preferred prey when food becomes limited. In addition, immunosupression due to starvation may promote parasitic infection and virulence.

An incidental finding of avian tuberculosis occurred in a Long-tailed Duck that died of trauma in the Beaufort Sea. There have been two reports of avian tuberculosis in captive sea ducks, two Common Eider in North America and one Hooded Merganser in Japan imported ten months earlier from Europe (Sato *et al.* 1996; Thornton *et al.* 1999). Avian tuberculosis is a chronic disease that generally occurs at low prevalence in wild birds in North America (Wobeser 1997; Friend 1999b). Its effect on wild bird populations is unknown but because *Mycobacterium avium* is also infectious to humans and domestic animals, infected wild birds represent a potential public health risk (Friend 1999b).

The absence of viruses as causes of mortality in sea ducks reported here may reflect a lack of knowledge of viruses in this group of waterfowl. Whilst sea ducks necropsied at the NWHC were tested for common viral pathogens of other waterfowl, no specific attempts were made to isolate other viruses that might have been present. Recent studies in the Wadden Sea and Baltic Sea in Europe indicate that viruses belonging to the families Parvoviridae, Reoviridae and Adenoviridae are important causes of mortality in sea ducks (Swennen 1991; Hollmén *et al.* 2002, 2003a,

unpublished observations). Adenoviruses also have been linked to mortality of long-tailed ducks in northern Alaska (Hollmén *et al.* 2003b). Although a species belonging to the genus *Avipoxvirus* caused the death of a Common Goldeneye in this study, there have been no other reports of mortality in sea ducks due to avian poxviruses. In addition, avian poxviruses rarely cause lesions sufficient to induce mortality in other waterfowl (Wobeser 1997; Hansen 1999). An avian poxvirus caused diphtheritic lesions of the mucosa and together with duck plague virus contributed to the mortality of American Black Duck (*Anas rubripes*) in New York in 1994 (Friend 1999c, NWHC unpublished observations).

The recent introduction of West Nile Virus (WNV) into North America has led to epornitic mortality in the American Crow (*Corvus brachyrhynchos*), although many other avian species in North America have been killed by the virus, including species of anatids (Anderson *et al.* 1999; Lanciotti *et al.* 1999; Steele *et al.* 2000; Bernard *et al.* 2001; Eidson *et al.* 2001; NWHC unpublished observations). West Nile Virus may not spread to subarctic areas in North America for the same reasons that it has not occurred in northern Europe, although it is enzootic in southern Europe (Hubálek and Halouzka 1999). Thus, it is likely that many sea ducks will not be exposed to WNV because they are found in subarctic areas during summer, when WNV transmission occurs in temperate regions (Bellrose 1976; Hayes 1989). However, WNV is likely to become enzootic in southern Canada during summer and because this area supports many breeding sea ducks, including the endangered Harlequin Duck, they should be monitored for WNV infection and possible effects of the virus (Health Canada 2002; Environment Canada 2003). To date we know of no published cases of mortality in sea ducks due to WNV.

Only three sea ducks that were submitted were killed by predators, two of these were nesting females. The importance of predation as a mortality factor is greatly underestimated by this type of study. Predation of breeding females has been suggested as a possible contributing factor in the decline of Spectacled Eiders on the Yukon-Kuskokwim Delta, Alaska (Stehn *et al.* 1993; Flint and Grand 1997).

Lead poisoning was the most frequently diagnosed toxicity identified in our review. Lead exposure and poisoning in waterfowl has been reported primarily as a result of ingested lead shot, and to a lesser extent from the ingestion of lead fishing weights and contaminated sediments (Sanderson and Bellrose 1986; Sears 1988; Sileo *et al.* 2001). Among sea ducks, lead poisoning with and without ingested lead shot has been diagnosed in the Common Eider, Spectacled Eider and Common Goldeneye, and ingested lead sinkers have been found in the Common Merganser, Red-breasted Merganser and White-winged Scoter (Clausen and Wolstrup 1979; Franson *et al.* 1995; Beyer *et al.* 1998; Hollmén *et al.* 1998; Sileo *et al.* 2001; Scheuhammer *et al.* 2003). Elevated blood lead concentrations have also been found in the Common Eider, Spectacled Eider and the Long-tailed Duck (Flint *et al.* 1997). Lead exposure in waterfowl causes anemia, loss of condition, and immunosuppression (Pain, 1989; Hohman *et al.* 1990; Rocke and Samuel 1991). These and other effects of lead exposure may contribute to population declines, or may impede recoveries of endangered populations, as elevated blood lead concentrations have been associated with reduced survival in the Canvasback (*Aythya valisineria*), American Black Duck, and Spectacled Eider (Samuel *et al.* 1992; Hohman *et al.* 1995; Grand *et al.* 1998). Lead shot for waterfowl hunting has been banned in the United States and several other countries (Fawcett and van Vessem 1995). However, sea ducks may continue to be exposed to lead in areas of the world without lead shot regulations and because of poor compliance or environmental factors resulting in the continued availability of lead shot in countries where it has been prohibited.

Oiling accounted for relatively few of the sea duck mortalities submitted to NWHC. It is possible that the frequency of petroleum toxicity is underrepresented because the obvious nature of oiled carcasses prevents biologists and natural resource managers from submitting them for necropsy examination in many instances. Recent evidence suggests that the effects of oil spills may continue for

years, resulting in chronic exposure of sea ducks that forage on benthic invertebrates (Peterson *et al.* 2003).

It is not surprising that firearm trauma was diagnosed in the Common Eider, Common Goldeneye, Bufflehead, Common Merganser, and Hooded Merganser, because hunting is permitted for these species. We also found gunshot as a cause of death in the Spectacled Eider and Steller's Eider, species sometimes taken by subsistence hunters (native Americans). In western Alaska, the estimated subsistence harvest of the Spectacled Eider was about 190 birds per year during 1995-1999, while estimates of Steller's Eider taken by subsistence harvest in the 1990s were considerably less (Petersen *et al.* 2000; Fredrickson 2001).

Starvation was an important mortality factor for adult sea ducks in this study and has the capability of causing large mortality if there are drastic changes in food supply. Extremely cold weather can cause widespread sea ice formation and reduce benthic food availability resulting in starvation if birds are too weak to seek warmer climates and alternative food supplies (Barry 1968; Baptist *et al.* 1997; Eric Taylor, personal communication). Large mortality events due to starvation occurred in the Common Eider in the winters of 1990/1991 and 1999/2000 in the Dutch Wadden Sea due to over fishing of bivalves by commercial fisheries (Beukema *et al.* 1998; Camphuysen *et al.* 2002).

Mortality of sea ducks can also be caused by entanglement and drowning in fishing nets as occurred in three Common Eider reported here. Methods to reduce the impact of fishing nets on vulnerable populations of sea ducks have been suggested (Fox *et al.* 1997). Whilst man made structures contributed to flight collisions in this study, sea ducks have also been reported to collide with the ground as well as man-made structures such as power transmission lines during periods of poor visibility (Leafloor *et al.* 1996; Mallory *et al.* 2001).

While some incidental findings are reported here, the primary objective was to document causes of mortality in sea ducks as determined by necropsy. These observations were opportunistic and therefore may not represent the most important long-term causes of mortality in sea duck populations. However, necropsies provide an opportunity to examine normal anatomical and physiological aspects of a species, such as its parasite fauna. We recommend that biologists consider these necropsy options because little is known about the biology of sea ducks. We also recommend that necropsy be routinely performed on sick and dead sea ducks to identify additional causes of morbidity or mortality. Information on recording data and handling and submitting specimens for necropsy during outbreaks of disease in sea ducks in North America is readily available (Friend and Franson 1999 available at http://www.nwhc.usgs.gov/pub_metadata/field_manual/field_manual.html; Davidson and Nettles 1997; Canadian Cooperative Wildlife Health Centre 2002). Although the proximate cause of mortality, such as predation, may appear obvious, necropsy may reveal underlying problems such as parasitism, infectious disease or exposure to contaminants, which could ultimately be responsible for the animal's death. One of the problems with necropsy as a technique for identifying causes of mortality and morbidity is that a low percentage of sick and dead wild birds are typically found (Wobeser 1994). Scavenging, rough terrain, adverse weather, the intensity of carcass searches, the tendency of sick wildlife to seek cover, carcass size and color, the state of decomposition, and camouflaging vegetative cover reduce carcass persistence and detection (Fowler and Flint 1997; Vyas 1999). Large numbers of dead animals are only found when mortality rates are high, so that normal scavenging mechanisms for removal of carcasses are overloaded (Wobeser 1994). In addition, many populations of sea ducks live in remote uninhabited areas where morbidity and mortality would not be observed (Bellrose 1976; Stehn *et al.* 1993). Therefore, it is important that systematic studies of the health of birds be conducted in isolated areas. For example, Hollmén *et al.* 2001 used serum biochemistries to examine health status of the Common Eider in the Baltic Sea. Finally, we suggest that the

mortality factors in sea ducks of North America are topics for new research efforts. Such research should include a focus on infectious organisms and naturally produced toxins because they have not been studied systematically in the past.

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